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Transcription of Rous sarcoma proviruses in rat cells is determined by chromosomal position effects that fluctuate and can operate over long distances.

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Most Rous sarcoma proviruses integrated in rat DNA are inactive. The active minority are not necessarily a consequence of long terminal repeat mutations and their transcription can fluctuate in parallel with previously demonstrated transitions in chromatin configuration. Transcriptional alterations cannot be attributed to variations in positive or negative factors that operate independently of the site of provirus integration. Moreover, distinct proviruses in the same cell can be differentially expressed and flanking cellular elements can act over several kilobases to inhibit provirus transcription. We postulate that position-dependent fluctuations in proviral repression can be mediated by trans-acting factors, and DNA transfections indicate that initiation and maintenance of this phenomenon are separable entities. Provirus activity presumably reflects otherwise inapparent cell controls that affect chromatin structure over long distances.

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